Naegleria fowleri

This article is about the protist. For the disease it causes, see Naegleriasis. For another protist with the same common name, see Balamuthia mandrillaris.

> This article **is missing information** about genome assemblies (drafts and one unpublished but finished genome on NCBI). Please expand the article to include this information. Further details may exist on the talk page. *(January 2021)*

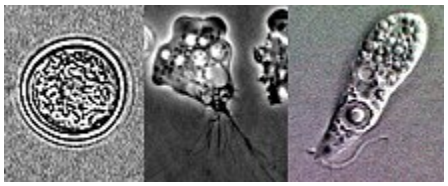
Naegleria fowleri, colloquially known as the **brain-eating amoeba**, is a species of the genus *Naegleria*. It belongs to the phylum Percolozoa and is technically classified as an amoeboflagellate excavate,[1] rather than a true amoeba. This free-living microorganism primarily feeds on bacteria but can become pathogenic in humans, causing an extremely rare, sudden, severe, and usually fatal brain infection known as naegleriasis or primary amoebic meningoencephalitis (PAM).[2]

Typically found in warm freshwater bodies such as lakes,[3] rivers, hot springs,[4] warm water discharge from industrial or power plants,[5] geothermal well water,[6] lakes and poorly maintained or minimally chlorinated swimming pools with residual chlorine levels under 0.5 mg/m^3,[7][8] water heaters,[9] soil, and pipes connected to tap water,[10] it can exist in either an amoeboid or temporary flagellate stage.[11]

Etymology

The organism was named after Malcolm Fowler, an Australian pathologist at Adelaide Children's Hospital, who was the first author of the original series of case reports of PAM.[12][13]

Life cycle



N. fowleri life-cycle stages, seen under a light microscope: cyst, trophozoite, flagellate (left to right).

Naegleria fowleri, a thermophilic and free-living amoeba, is primarily found in warm and hot

freshwater environments such as ponds, lakes, rivers, and hot springs.[14] As temperatures rise, its population tends to increase. Although the amoeba was initially identified in Australia in the 1960s, it is believed to have evolved in the United States.[15] *N. fowleri* exists in three forms: cyst, trophozoite (ameboid), and biflagellate. While it does not form cysts in solid human tissue, where only the amoeboid trophozoite stage is present, the flagellate form has been discovered in cerebrospinal fluid.

Cyst stage

To endure harsh environmental conditions, trophozoites transform into microbial cysts,[16] spherical, single-layered structures about 7–15 μm in diameter, enclosing a single cell nucleus.[17] Acting as a resilient capsule, the cyst enables the amoeba to withstand adverse circumstances. Factors triggering cyst formation include food scarcity, overcrowding, desiccation, waste accumulation, and cold temperatures. When conditions improve, the amoeba can emerge through the pore or ostiole at the center of the cyst. *N. fowleri* has been observed to encyst at temperatures below 10 °C (50 °F).[17][16]

Trophozoite stage

The trophozoite stage is the infective phase for humans, during which the organism can actively feed and replicate. The trophozoite attaches to the olfactory epithelium, follows

Naegleria fowleri

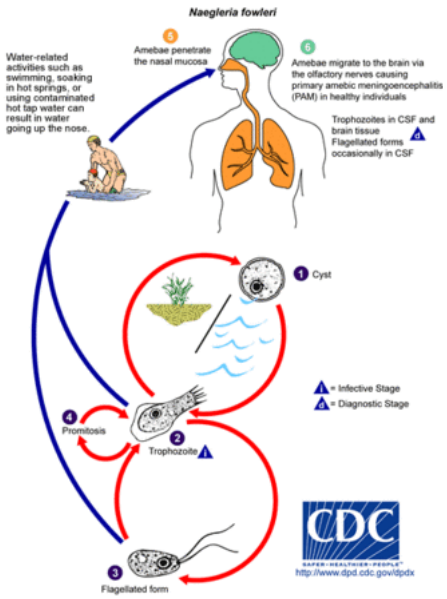
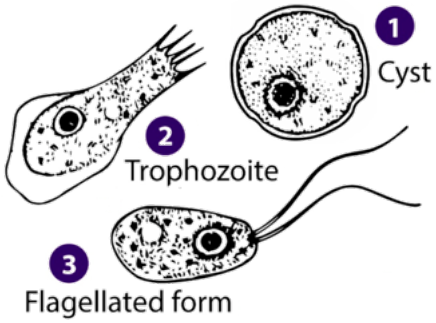


Diagram depicting the stages of *Naegleria fowleri*'s life-cycle and environment at that stage



Drawings of the three stages *Naegleria fowleri*'s life-cycle

Scientific classification

Domain:	Eukaryota
Phylum:	Percolozoa
Class:	Heterolobosea
Order:	Schizopyrenida
Family:	Vahlkampfiidae
Genus:	*Naegleria*

Species:	***N. fowleri***
Binomial name	
Naegleria fowleri Carter (1970)	

the axons of olfactory receptor neurons through the cribriform plate in the nasal cavity, and enters the brain. This reproductive stage of the protozoan organism transforms around 25 °C (77 °F), and thrives best at approximately 42 °C (108 °F), multiplying through binary fission.

Trophozoites are characterized by a nucleus surrounded by a flexible membrane. They move via pseudopodia, extending parts of their cell membrane (pseudopods) and filling them with protoplasm to facilitate locomotion. Pseudopods form in the direction of movement. In their free-living state, trophozoites feed on bacteria. In tissues, they appear to phagocytize (enclose and digest) red blood cells and cause tissue damage either through the release of cytolytic substances or by direct cell-to-cell contact using cytolytic membrane proteins.[17]

As trophozoites, *Naegleria fowleri* may develop approximately 1 to 12 structures on their membrane known as amoebastomes (amorphous cytostomes), also referred to as "suckers" or "food cups," which they use for feeding in a manner similar to trogocytosis.[18]

Flagellate stage

The flagellate stage of *Naegleria fowleri* is pear-shaped and biflagellate (with two flagella). This stage can be inhaled into the nasal cavity, typically during activities such as swimming or diving. The flagellate form develops when trophozoites are exposed to a change in ionic strength in the fluid it is in, such as being placed in distilled water. The flagellate form does not exist in human tissue, but can be present in the cerebrospinal fluid. Once inside the nasal cavity, the flagellated form transforms into a trophozoite within a few hours.[17]

Ecology

Naegleria fowleri, an excavata, inhabits soil and water. It is sensitive to drying and acidic conditions, and cannot survive in seawater. The amoeba thrives at moderately elevated temperatures, making infections more likely during summer months. *N. fowleri* is a facultative thermophile, capable of growing at temperatures up to 46 °C (115 °F).[11] Warm freshwater with

an ample supply of bacteria as food provides a suitable habitat for amoebae. Locations where many amoebic infections have occurred include artificial bodies of water, disturbed natural habitats, areas with soil, and unchlorinated or unfiltered water.

N. fowleri appears to flourish during periods of disturbance. The "flagellate-empty" hypothesis suggests that Naegleria's success may stem from decreased competition when thermosensitive protozoal fauna do not survive changes in temperature. In other words, *N. fowleri* thrives when other predators consuming its food supply are absent. This hypothesis implies that human disturbances, such as thermal pollution, increase the abundance of *N. fowleri* by eliminating its resource competitors. Amoeboflagellates have a motile flagellate stage that aids in dispersal, that is advantageous in environments cleared of competing organisms.

Pathogenicity

Main article: Naegleriasis

Roman Baths in Bath, England, closed for bathing since 1978 due to the presence of *N. fowleri*.

N. fowleri may cause a usually fatal infection of the brain called naegleriasis, primary amoebic meningoencephalitis (PAM), amoebic encephalitis/meningitis, or simply *Naegleria* infection. Infections most often occur when water containing *N. fowleri* is inhaled through the nose (aspirated), where it then enters the nasal and olfactory nerve tissue, travelling to the brain through the cribriform plate.[19] Swallowing contaminated water does not cause infection by *N. fowleri*.[20] Infections typically occur after swimming in warm-climate freshwater, although there have been cases in cooler climates such as Minnesota, US.[21] In rare cases, infection has been caused by nasal or sinus rinsing with contaminated water in a nasal rinsing device such as a neti pot.[10]

N. fowleri normally eat bacteria, but during human infections, the trophozoites consume

astrocytes and neurons. The reason why *N. fowleri* passes across the cribriform plate is not known, but the neurotransmitter acetylcholine has been suggested as a stimulus precipitating the action, as a structural homolog of animal CHRM1 has been shown to be present in *Naegleria* and *Acanthamoeba*.[22]

It takes one to twelve days, median five, for symptoms to appear after nasal exposure to *N. fowleri* flagellates.[23] Symptoms may include headache, fever, nausea, vomiting, loss of appetite, altered mental state, coma, drooping eyelid, blurred vision, and loss of the sense of taste.[24] Later symptoms may include stiff neck, confusion, lack of attention, loss of balance, seizures, and hallucinations. Once symptoms begin to appear, the patient usually dies within two weeks. *N. fowleri* is not contagious; an infected person cannot transmit the infection. From 2013 to 2022, 29 infections were reported in the US, which compares with about 4,000 annual deaths by drowning.[25] It is so rare that individual cases are often reported internationally.[26]

Animals may be infected by *Naegleria fowleri*. This is rarely observed, although it may occur and be overlooked. Experimentally, mice, guinea pigs, and sheep have been infected, and there have been reports of South American tapirs and cattle contracting PAM.[27]

Treatment

Main article: Naegleriasis § Treatment

The core antimicrobial treatment consists of the antifungal drug amphotericin B,[28] which inhibits the pathogen by binding to its cell membrane sterols, causing cell membrane disruption and pathogen death;[29] however, even with this treatment, the fatality rate is greater than 97%.[25][30] New treatments are being sought.[25][31] Miltefosine, an antiparasitic drug that inhibits the pathogen via disrupting its cell survival signal pathway PI3K/Akt/mTOR,[29] has been used in a few cases with mixed results.[32]

A key factor to effective treatment is the speed of diagnosis. Naegleriasis is rare, and is often not considered as a likely diagnosis; therefore, the clinical laboratory's identification of the microorganism may be the first time an amoebic etiology is considered. Rapid identification can help to avoid delays in diagnosis and therapy. Amoeba cultures and real-time polymerase chain reaction (PCR) studies for *N. fowleri* are diagnostic of PAM, but they are not readily available at most institutions, and would have to be carried out at a reference laboratory. The time of presentation of the patient may affect the identification of the microorganism also, as PAM has an incubation period ranging from 1 to 12 days.[25] The clinical signs of PAM are similar to

bacterial and viral meningitis, including fever, neck stiffness, and severe headaches. Symptoms can progress to prolonged nausea, vomiting, and even seizures. The disease can progress to acute hemorrhagic necrotizing meningoencephalitis. After symptoms start the patient typically dies within 1 to 18 days, typically about 5 days.[25] A variable delay in treatment can be secondary to time intervals in multiple stages of care, including exposure to exhibition of symptoms; arrival for treatment at a health care facility; workup of the diagnosis (initial diagnosis of likely bacterial meningitis); and finally, from diagnosis to initiation of recommended therapy. Successful treatment of PAM is rare; treatment can only be attempted after correct diagnosis, which relies on rapid recognition of the microorganism by medical technologists and pathologists. It is critical that medical technologists consistently provide timely CSF evaluation, explore the diagnosis of PAM, and look for amoebae in the setting of meningitis, especially in summer.[33]

Cases in Pakistan

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In Pakistan, the number of reported cases has surpassed the global total due to insufficient healthcare infrastructure and limited awareness of Naegleria fowleri. As a result, only a small fraction of cases are correctly identified as Primary Amebic Meningoencephalitis (PAM), with the majority of cases misdiagnosed as viral meningitis.

For the very first time in Pakistan, *N. fowleri* genotype has been identified as type-2. Phylogenetic analysis showed that *N. fowleri* isolate from Pakistan is among the latest descendants, i.e., evolved later in life.[34]

See also

- *Acanthamoeba* – an amoeba that can cause amoebic keratitis and encephalitis in humans
- *Balamuthia mandrillaris* – an amoeba that is the cause of (often fatal) granulomatous amoebic meningoencephalitis
- *Entamoeba histolytica* – an amoeba that is the cause of amoebiasis, or *amoebic dysentery*
- *Leptospira* – a zoonotic bacteria that causes leptospirosis

- Methicillin-resistant *Staphylococcus aureus* (MRSA)
- Necrotizing fasciitis – the "flesh-eating disease", caused by certain types of bacteria
- *Toxoplasma gondii* – cat-carried protozoan that causes the disease toxoplasmosis
- *Vibrio vulnificus* – warm saltwater infectious bacteria

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External links

- *Naegleria* information site (https://www.cdc.gov/parasites/naegleria/) from the Centers for Disease Control and Prevention

Wikimedia Commons has media related to ***Naegleria fowleri***.

- *Naegleria* (http://tolweb.org/Naegleria/124653) from The Tree of Life Web Project